California MEDICINE
OFFICIAL JOURNAL OF THE CALIFORNIA MEDICAL ASSOCIATION

Volume 97 AUGUST 1962 Number 2

Correctable Cardiac Failure

ARTHUR SELZER, *M.D.*, and FRANK GERBODE, *M.D.*, San Francisco

• **The concept of reversible cardiac failure has hitherto been applicable mostly to rare instances of acute afflictions of the myocardium wherein cardiac compensation returns with the healing of the process. Recent strides in cardiac surgery have brought into focus a wide variety of conditions where operative removal of the cause of heart failure can successfully restore compensation.**

The concept of increased work of the heart—cardiac overload—is presented and classified with special reference to those forms where surgical removal of the cause of the overload is possible.

Now, since surgical treatment of a patient in functional class IV need no longer entail risk of prohibitive mortality, a careful search is indicated in patients in a state of chronic cardiac failure, particularly in the younger age group, for a correctable factor or factors.

IN SPITE OF DRAMATIC ADVANCES in the treatment of hypertensive diseases, the term "curable" hypertension can only be applied to the relatively small group of cases in which the cause of hypertension can be removed surgically, such as coarctation of the aorta, pressor-substance producing tumors, or correctable renal and renal-vascular disorders. Other forms of hypertension can at best be controlled by continuous therapy. An analogous situation has been created by recent surgical strides in the treatment of chronic cardiac failure, for in a growing number of conditions the cause of failure can be removed surgically, reversing otherwise intractable heart failure.

There are few concepts in clinical medicine that match cardiac failure with regard to the controversy and confusion surrounding its clinical, physiological and metabolic definition. The introduction of modern, precise physiological methods into clinical medicine helped to clarify many points but at the same time brought into focus other unanswered questions. However, two concepts can be considered as generally accepted: (a) the two ventricles can fail and recover from failure independently of each other; (b) heart failure is usually caused by increased work—"overload" of a cardiac ventricle.

The work of the heart is expressed in physiological terms as a product of the quantity of blood ejected into the arterial system and the pressure against which it is expelled. It follows that overloading of a ventricle can occur when the output is excessive or the pressure elevated. Physiological increase in cardiac work occurs during exercise, when cardiac output rises; during stress and excitement when pressure is increased. Pathological cardiac overwork occurs when pressure overload or volume overload occurs continuously rather than intermittently. Such pathological overload stimulates a compensatory increase in muscle mass—cardiac hypertrophy—which can, for variable lengths of time, maintain an adequate circulation in spite of its overloading. Eventually the hypertrophied ventricle becomes inefficient and the symptom complex

From the Institute of Medical Sciences, Presbyterian Medical Center, San Francisco 15.

Presented at the Eighth Annual Symposium on Cardiovascular Diseases of the Monterey County Heart Association, Fort Ord, January 20, 1962.

Submitted March 5, 1962.

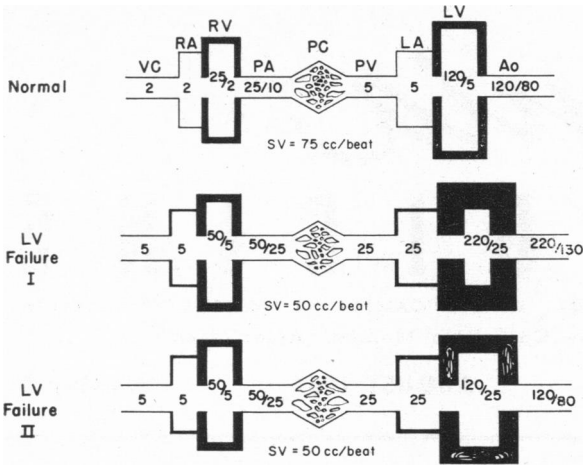

Figure 1.—Diagrams of the circulation: top, normal; middle, left ventricular failure due to hypertensive overload; bottom, that due to myocardial scars. The top drawing presents a diagram of a normal circulatory system by indicating pressures in the various regions and a normal stroke volume of 75 cc. per beat. The middle drawing presents left ventricular failure due to absolute systolic overload of chronic hypertension. Left ventricular hypertrophy is shown and failure of that ventricle indicated by an elevation of the diastolic pressure to 25 mm. and by reduced stroke volume to 50 cc. per beat. Left ventricular diastolic hypertension leads to elevation of left atrial pressure and passive pulmonary hypertension. However, the right ventricle, though overloaded, is still competent. The bottom drawing presents left ventricular failure due to relative left ventricular overload from chronic scarring of that ventricle after myocardial infarction. Left ventricular hypertrophy is also present here and its hemodynamic sequelae are identical with those shown above.

of chronic cardiac failure develops. A variety of cardiac diseases exemplify conditions associated with chronic ventricular overload. Hypertension and valvular stenosis produce pressure overload; volume overload is brought about by valvular regurgitation, shunting lesions and hypercirculatory states. Pressure overload, often referred to as *systolic* overload, causes concentric ventricular hypertrophy unassociated with dilatation until later, during the stage of decompensation. Volume overload, as *diastolic* overload, leads to early development of cardiac dilatation (eccentric ventricular hypertrophy).

The concept of chronic overloading of the circulation is applicable to many but not all forms of cardiac failure. The most important form of chronic cardiac failure in which the work of the heart appears not to be increased is that occurring in the late stage of ischemic heart disease ("arteriosclerotic heart disease"). Yet in such cases chronic failure is likely to occur only if a significant portion of the left ventricular myocardium is replaced by functionally inactive scar tissue. It follows that the normal cardiac work has to be performed by a considerably reduced number of muscle fibers and that therefore the work *per muscle fiber* is increased. One is thus justified in accepting this condition as a variant of the chronic overload and in using the term "relative cardiac overload."

A comparison of "absolute" and "relative" overloading of the left ventricle is presented in diagrammatic form in Figure 1.

There are, however, conditions in which the concept of overloading of a cardiac ventricle as a cause of cardiac failure does not seem to be applicable. Among them are the various inflammatory, degenerative or toxic conditions affecting cardiac performance, in which all or most muscle fibers are uniformly affected. Many of these afflictions are acute and constitute truly reversible cardiac failure: the circulatory derangement returns to normal with the healing of the myocardial lesion.

When can chronic cardiac failure be considered reversible? The mere disappearance of clinical manifestations of heart failure does not provide evidence of reversibility. Studies in our laboratory[1] have demonstrated that patients with various forms of cardiac failure may become totally asymptomatic in response to therapy and yet almost always show abnormal hemodynamic findings, demonstrating that they are merely in a state of *controlled* heart failure. The only real examples of reversibility of chronic cardiac failure are those in which chronic overload responsible for failure can be totally or partially eliminated. It is obvious that only absolute cardiac overload can be eliminated: relative overload, as stated above, is caused by reduction of the number of contracting myocardial units and is not correctable.

A simple example of the elimination of cardiac overload is the treatment of hyperthyroidism or anemia. Both of these conditions lead to a hypercirculatory state but seldom of themselves lead to chronic cardiac failure, acting mostly as a contributory factor in combined cardiac lesions. Therefore, it is quite rare to find spectacular elimination of chronic heart failure by the correction of these states. It is by surgical elimination of overload that dramatic elimination of advanced heart failure and a complete rehabilitation of a chronic "cardiac cripple" can be brought about. The following is a brief discussion of the more important forms of surgically correctable cardiac overload.

Left ventricular failure

Systolic overload of the left ventricle. This is responsible for heart failure after prolonged systolic left ventricular hypertension, which in turn may be caused by either *arterial hypertension* or by *aortic stenosis*. Chronic cardiac failure from arterial hypertension is seldom reversible unless a surgically curable form of hypertension exists. The fact that medical treatment of hypertension usually does not

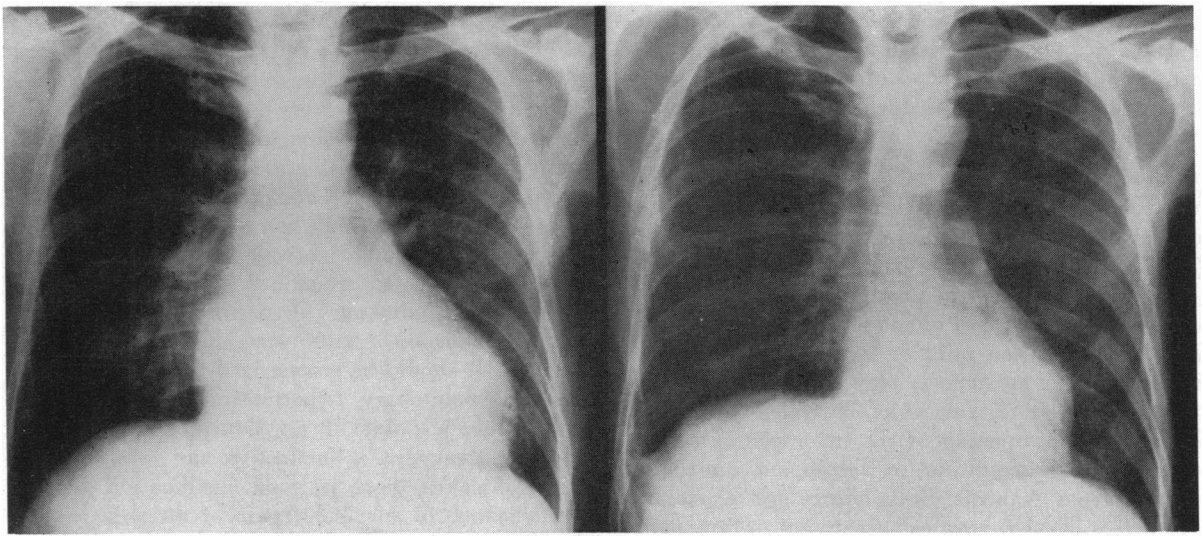

Figure 2.—Anteroposterior roentgenogram of a patient with severe mitral regurgitation: left, before; right, after mitral valvular repair.

reverse cardiac failure resulting from it is probably related to the widespread vascular deterioration which is usually present by the time chronic cardiac failure has developed.

On the other hand, aortic stenosis may lead to intractable cardiac failure which is quickly and dramatically reversed if the obstruction can be entirely relieved, therefore restoring normal left ventricular systolic pressure.

Diastolic overload of the left ventricle. The two common conditions with increased cardiac work of this type are *patent ductus arteriosus* and *aortic regurgitation.* In both, the left ventricle ejects an excessive amount of blood. In both, hypertrophy associated with early dilatation of that chamber occurs. Left ventricular overload of this type is relatively well tolerated, and it is not unusual to find an extreme degree of cardiomegaly with little or no disability. Cardiac failure occurs late but is often then unrelenting, and refractory to therapy, unless surgical treatment can eliminate the overload altogether. The dramatic reversal of cardiac failure and reduction in cardiac size in cases of this category in which surgical treatment was successful are among the most spectacular results in the field of cardiac surgery.

Combined ventricular failure

Left ventricular failure from any cause produces passive pulmonary hypertension, as shown in Figure 1, which leads to systolic overload of the right ventricle. This is occasionally exaggerated by the occurrence of pulmonary arteriolar spasm or pulmonary vascular disease developing secondarily to passive pulmonary hypertension. Thus it is well known that left ventricular failure is the commonest cause of right ventricular failure which eventually results from right sided overload. However, two conditions are known to cause initial overload of both ventricles: *mitral regurgitation* and *ventricular septal defect.* Mitral regurgitation produces diastolic overload of the left ventricle by necessitating the ejection of large quantities of blood into the atrium in addition to that into the aorta.

Recent studies have shown that as much as 75 per cent of the total output may be regurgitated into the atrium. However, in contrast to aortic regurgitation and patent ductus, mitral regurgitation causes left atrial hypertension. Consequently the resulting right ventricular overload occurs *before* the onset of left ventricular failure. Ventricular septal defect associated with a sizable left-to-right shunt leads to increased output of both ventricles, causing diastolic overloading of both sides. In both ventricular septal defect and mitral regurgitation, left ventricular overload and failure usually predominate, but pulmonary hypertension may progress to a point where right ventricular hypertrophy and failure come into the foreground. Surgical therapy may completely eliminate the overload and reverse cardiac hypertrophy and failure. Figure 2 illustrates the regression of cardiomegaly in a patient who before surgical treatment was totally disabled by combined right and left heart failure due to mitral regurgitation and who became virtually asymptomatic after successful repair of the valve lesion.

Right ventricular failure

Right ventricular failure is the result of right ventricular overload under conditions analogous to events in the left heart referred to above. However, only absolute right ventricular overload is

of importance, since involvement of the right ventricular myocardium leading to its relative overload is not known to be of major importance. Again, two forms of right sided overload can occur: pressure and volume.

Systolic overload of the right ventricle. Increased work of the right ventricle here is caused by elevated systolic pressure in it, which may be due to abnormally high resistance at the pulmonary valve or beyond it; pulmonary stenosis and pulmonary hypertension. *Pulmonary stenosis* is usually well tolerated when mild or moderate in severity. Severe pulmonary stenosis may lead to intractable right ventricular failure, which is occasionally complicated by the opening of the incompletely sealed foramen ovale, resulting in significant anoxemia or even gross cyanosis. Both failure and anoxemia are reversible by surgical treatment. *Pulmonary hypertension* occurs in a variety of conditions and has many causes. In general, pulmonary hypertension falls into three categories: increased resistance within the pulmonary vascular tree, increased resistance beyond the pulmonary venous system ("passive pulmonary hypertension"), and the combination of both. The first type is, as a rule, irreversible. The other two may or may not be reversible; passive pulmonary hypertension may be caused by left ventricular failure or by mitral valve disease. The former has already been discussed: its reversibility depends on whether or not the left sided lesion is correctable. *Mitral stenosis* is the commonest and the best known cause of reversible passive pulmonary hypertension. It should be noted, however, that uncomplicated mitral stenosis, even severe, seldom causes severe enough pulmonary hypertension to lead to chronic right ventricular failure, which if present is almost always due to the development of secondary pulmonary vascular reactions. Thus, in some 15 per cent of cases of mitral stenosis, pulmonary hypertension of a combined type develops, which is then apt to cause chronic right ventricular failure. Two points are noteworthy in connection with mitral stenosis: pulmonary hypertension complicating mitral stenosis may become severe enough to dominate the clinical picture and obliterate the clinical landmarks identifying mitral stenosis. Serious errors have been made on occasion by mistaking such cases of mitral stenosis for irreversible primary pulmonary hypertension. Furthermore, surgical correction of mitral stenosis abolishes not only passive pulmonary hypertension but leads to gradual regression of pulmonary vascular disease, thus further accentuating beneficial effects of the operation.[2]

Diastolic overload of the right ventricle is exemplified by the *atrial septal defect.* In the presence of an interatrial communication the lower resistance to diastolic filling of the right ventricle causes large left-to-right shunt with the volume load of the right ventricle as high as four times that of the left. Curiously, severe volume overload of the right ventricle is very well tolerated and cardiac failure from an uncomplicated atrial septal defect is virtually unknown in children and adolescents. In adults, however, the chronic increase in pulmonary blood flow often leads to secondary pulmonary vascular disease and pulmonary hypertension. When that happens, chronic right ventricular failure may develop. It should be emphasized that unlike mitral stenosis, pulmonary hypertension in atrial septal defect has a point of no return, beyond which surgical treatment is ineffective and may even be harmful. Thus there is good justification for the performance of surgical repair of atrial septal defect in asymptomatic patients, provided facilities are available for the performance of such operations with a minimum mortality.

As stated, the foregoing discussion dealt only with the more common forms of cardiac overload leading to chronic cardiac failure. There are many other rarer forms of heart disease, some of which are reversible by surgical treatment, some not. It is particularly noteworthy that cardiac lesions frequently occur in combinations. Both congenital heart disease and rheumatic heart disease, the two principal etiologic causes of correctable cardiac lesions, show a tendency to affect more than one area of the heart. The hemodynamic consequences of combined lesions are usually additive and physiological studies often can unravel the respective contribution of the component lesions to cardiac disability. It is of considerable importance to distinguish between the principal overloading lesion and the lesser contributory factors, for to attempt surgical repair of all the anatomical defects might seriously and unnecessarily increase the risk. It is necessary to appreciate the fact that, if chronic cardiac failure develops because the heart is overloaded by 150 per cent over its normal work, a complete reversal of failure may result by the reduction of the overload to 25 or 50 per cent. A partial correction, wherein one lesion is repaired and another left untreated, is indicated when the total repair seriously magnifies the surgical hazard. Such situations develop frequently in the treatment of combined mitral and aortic lesions where the mitral disease predominates, or in atrial septal defects complicated by one or two transposed pulmonary veins, to name two examples.

Among unusual problems associated with surgical treatment of cardiac failure, two deserve comment. The first, constrictive pericarditis, does not cause heart failure in the ordinary sense, but rather congestive phenomena masquerading as heart

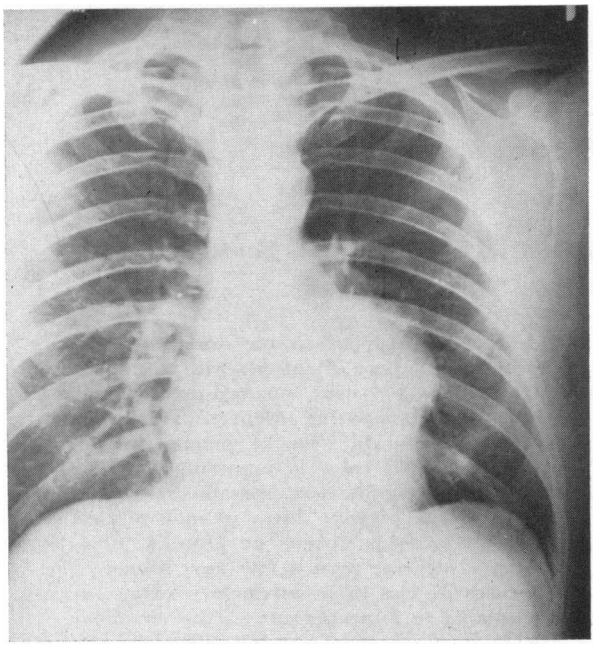

Figure 3.—Anteroposterior roentgenogram of a patient with a large aneurysm of the left ventricle.

failure. Surgical treatment of constrictive pericarditis, the oldest operation in the field of cardiac surgery, is probably performed less often than it ought to be because less typical forms of it may be overlooked. The classical picture of congestive "failure" with a small and quiet heart is not always present, and the differentiation between constrictive pericarditis and conventional forms of cardiac failure may be very difficult. The second problem concerns ventricular aneurysms. As the resection of aneurysms of the heart becomes technically possible and relatively safe, it is often tempting to consider the mere presence of such aneurysms as indication for operation. Figure 3 is a roentgenogram, taken in 1945, showing a large aneurysm of the left ventricle. The patient now is well and virtually asymptomatic. Unlike aneurysms of the great vessels, healed aneurysms virtually never rupture. Physiologically they resemble mitral regurgitation in that during systole the left ventricle has to eject blood into the aorta and also fill the aneurysmal sac, which overloads that cardiac chamber. As in mitral regurgitation, such left ventricular overload may be small, causing no major circulatory derangement, or large, leading to heart failure. Thus if chronic failure is present in a case of ventricular aneurysm and if the systolic expansion of the aneurysm indicates loss of large volume of blood, surgical treatment is indicated and often leads to spectacular improvement.

What is the role of the practicing physician in relation to the problem of surgical treatment of cardiac failure? Using again the example of hypertension, it is now generally recognized that every case of severe hypertension, particularly in a younger person, should be investigated with regard to the possibility of a curable form of hypertension. A similar attitude is justified in dealing with chronic cardiac failure. Only a high index of suspicion regarding reversibility of chronic cardiac failure will bring a satisfactory yield of surgically correctable cases. It might seem, superficially, that such cases are easy enough to recognize that they should present no diagnostic problem. While this is true for a typical case of mitral stenosis, aortic stenosis or patent ductus arteriosus, there are many instances in which the correct diagnosis is exceedingly difficult to make and may require the most complex laboratory procedures. It is well to remember that the very presence of cardiac failure or of the complications that enhance failure, such as pulmonary hypertension, brings into the picture atypical and confusing clinical features. It has already been mentioned that pulmonary hypertension complicating mitral stenosis often suggests the erroneous diagnosis of primary pulmonary hypertension. Pulmonary hypertension tends to obliterate the characteristic murmurs of patent ductus arteriosus and ventricular septal defect. Cardiac failure may reduce the murmur of aortic stenosis to a seemingly inconsequential systolic murmur. Severe cardiac failure may lower the wide pulse pressure of aortic regurgitation to a point where it is no longer suspected as being the principal cause of the cardiac overload. Apical systolic murmurs are exceedingly common in any form of left ventricular failure and occasionally it is difficult to decide whether mitral insufficiency is the cause or the effect of cardiac failure.

In selecting patients with cardiac failure for surgical treatment, it is essential that a comprehensive diagnostic evaluation be made. This includes not only an analysis of the hemodynamic factors leading to cardiac overload but a search for such complicating factors as coronary artery disease, which, if demonstrated in the coronary angiogram, would make an operation inadvisable. The risk of operating upon properly selected patients in chronic failure is higher than average, but not prohibitive in the hands of a team with proper experience and facilities to handle such cases.

Presbyterian Medical Center, Clay and Webster Streets, San Francisco 15 (Selzer).

REFERENCES

1. Selzer, A., and McCaughey, D. J.: Hemodynamic patterns in chronic cardiac failure, Am. J. Med., 28:337, 1960.

2. Selzer, A., and Malmborg, R. O.: Some factors influencing pulmonary vascular resistance in mitral valvular disease, Amer. J. Med., 32:532, 1962.